

## Event Abstract

# A Fast-Slow Minimal Model for Medium Spiny Neurons: A Geometrical Perspective

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The role of basal ganglia in motor control is well-known [1,2], but it also takes part in high order cognitive processes, such as reward-related learning, goal-directed behavior or selective attention [3-5]. The complicated and creative information processing ability of basal ganglia makes it home for decisions amongst available competitive choices. Dysfunction of this sub-cortical network, along with the related neurotransmitters (e.g. dopamine), causes neurodegenerative diseases as Parkinson's Disease or Huntington's Disease [6,7]. D1 and D2 type dopamine receptors have a modulatory effect on this network by controlling the GABAergic signalling from the basal ganglia main input station, striatum, to the direct and indirect pathways. The striatal medium spiny neurons (MSNs) play key role in the formation of the antagonistic functions of direct and indirect pathways. We focused on the state-space behavior of the conductance-based computational model of MSNs which is constituted with nonlinear dynamical systems' approach.

The typical behavior of the striatal MSNs is bursting activity as claimed in [8] and these neurons play a role in synaptic plasticity. Since the conventional Hodgkin-Huxley neuron model is not suitable for modelling the striatal MSNs, L-type Ca<sup>2+</sup> (high threshold calcium), Kv1.2-containing K<sup>+</sup>, Calcium activated Calcium and Calcium activated Potassium (afterhyperpolarization) ion channels are also considered in connection with the role of dopamine receptors in MSNs. The bursting activity that is appeared in torus canards [9] of the proposed MSNs minimal model is explained via the qualitative theory of fast-slow dynamical systems with a significant ratio of time scale parameter ( $\epsilon \ll 1$ ). In the model we proposed, the generation of bursting pattern depends on two different current mechanisms. Slow current system (sodium) is responsible for the bifurcation branch between equilibrium point and the limit cycles (Andronov-Hopf Bifurcation) by effecting the fast current system (afterhyperpolarization). At the same time, fast current system has a role in spiking activity of the bursting patterns (saddle-node of periodic orbits). A torus bifurcation also appears in the full system. The Subhopf/Fold Cycle type bursting activity of the proposed model is an example of Ca<sup>2+</sup> gated inactivation of an inward current [10] and it depends on fold limit cycle bifurcation. Besides codimension-1 bifurcations, period doubling and torus bifurcation are also observed with numerical continuations.

The proposed model is capable of explaining how dopamine release modulates the functions of

striatum. The geometry of the phase portraits, imported from significant bifurcation points, allow us to understand the ability of the proposed model. Thus, the whole architecture of the proposed model is shown to be captured by bifurcation analysis.

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## Conflict of Interest

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

## References

- [1] Gurney, K., Prescott, T. J. and Redgrave, P., 2001. Computational Model of Action Selection in the Basal Ganglia I: A New Functional Anatomy, *Biological Cybernetics*, 84(6) pp. 401-410.
- [2] Houk, J.C., Bastianen, C., Fansler, D., Fishbach, Fraser, D., Reber, P.J., Roy, S.A, and Simo, L.S., 2007. Action selection and refinement in subcortical loops through basal ganglia and cerebellum, *Phil. Trans. R. Soc. B.*, 362(1485), pp. 1573-1583.
- [3] Cohen, M.X. and Frank, M.J., 2009. Neurocomputational models of basal ganglia function in learning, memory and choice, *Behavioural Brain Research*, 199, pp. 141-156.
- [4] Dayan, P. and Balleine, B.W., 2002. Reward, motivation, and reinforcement learning, *Neuron*, 35(2), pp. 285–298.
- [5] Hollerman, J.R., Tremblay, L. and Schultz, W., 2000. Involvement of basal ganglia and orbitofrontal cortex in goal-directed behavior, *Progress in Brain Research*, 126, pp. 193–215.
- [6] Wiecki, T.V. and Frank, M.J., 2010. Neurocomputational models of motor and cognitive deficits in Parkinson’s disease, *Progress in Brain Research*, 183, pp. 275–297.
- [7] Montoya, A., Price, B.H., Menear, M. and Lepage, M., 2006. Brain imaging and cognitive dysfunctions in Huntington’s disease, *Journal of Psychiatry Neuroscience*, 31(1), pp. 21–29.
- [8] Izhikevich E.M., Desai N.S., Walcott E.C. and Hoppensteadt F.C.: Bursts as a unit of neural information: selective communication via resonance. *Trends Neurosci* 2003, 26, pp. 161–167.
- [9] Burke, J., Desroches, M., Barry, A.M., Kaper, T.J. and Kramer, M.A., 2012. A showcase of torus canards in neuronal bursters, *The Journal of Mathematical Neuroscience*, 2(3).
- [10] Izhikevich, E.M., 2007. *Dynamical Systems in Neuroscience, The Geometry of Excitability and Bursting*, The MIT Press.